

Nonconvulsive Status Epilepticus Causing Prolonged Stupor After Intraventricular Hemorrhage: Report of a Case

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Key Words

Elderly Patients
Electroencephalography
Intraventricular Hemorrhage
Nonconvulsive Status Epilepticus
Stupor

ABSTRACT

We describe the case of an octogenarian woman who experienced a severe alteration of mental state due to non-convulsive status epilepticus (NCSE) complicating an intraventricular hemorrhage. Our report emphasizes that NCSE may be the cause of unexplained neurological deterioration in elderly patients with acute brain injury.

INTRODUCTION

Nonconvulsive status epilepticus (NCSE) is a well-known cause of altered mental state whose diagnosis may be particularly difficult in elderly people.¹ Cerebrovascular disease is considered a frequent etiology of epilepsy in older patients, however, intraventricular hemorrhage is an unreported condition associated with NCSE.

CASE REPORT

An 88-year-old woman was admitted to our hospital because of acute alteration of consciousness, nausea and vomiting. She had antecedents of hypertension and had suffered from an ischemic infarction in the territory of the left middle cerebral artery that had caused a mild right hemiparesis. On examination, she was slightly confused, oriented in person and disoriented in time and place. Her speech was not fluent and there was a discrete weakness of the right limbs. Plantar responses were flexor. A computed tomography (CT) scan of the brain revealed a left intraventricular hemorrhage and a small hematoma localized in the left thalamus (Figure 1).

During the next 72 hours, her level of consciousness worsened dramatically, and she became stuporous. Although the clinical worsening was thought secondary to rebleeding, cerebral edema or hydrocephalus, an electroencephalogram (EEG) was requested. The EEG showed the existence of continuous epileptiform discharges involving the entire left hemisphere in keeping with the diagnosis of localization-related (partial) NCSE (Figure 2a). Despite the

onset of treatment with intravenous phenytoin (100 mg/8 h), the clinical state of the patient was unmodified for the next 3 days. Three consecutive EEGs carried out over those days revealed generalized continuous epileptiform activity compatible with a secondarily generalized NCSE (Figure 2b).

The patient's metabolic status throughout the course of the disease was carefully controlled, and particular interest was paid to avoid metabolic disturbances such as hyponatremia or hypokalemia, which are known predisposing factors of NCSE. Antiepileptic therapy with valproate (1000 mg/24 h) and clonazepam (1.0 mg/24h) was added to her previous treatment. On the following 17 days, her level of consciousness was fluctuant ranging from somnolence to profound stupor. Several EEGs showed the persistence of ongoing generalized epileptiform discharges during these days. On day 23 after admission, she was conscious and able to understand simple commands. At that time, an EEG disclosed features compatible with a moderate diffuse encephalopathy and resolution of NCSE (Figure 2c). Finally, she was discharged to another hospital for chronic pathology.

DISCUSSION

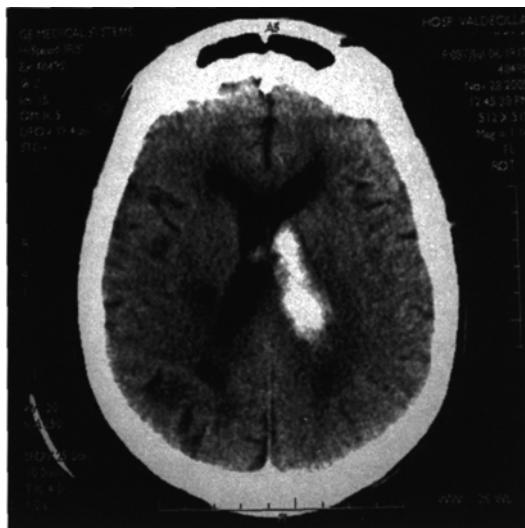
Intraventricular hemorrhage is a rarely described cause of NCSE.^{2,3} Recently, Dennis et al.³ carried out the first investigation studying the frequency and clinical features of NCSE among comatose patients with subarachnoid hemorrhage. Eight out of 26 patients who underwent continuous electroencephalographic monitoring were diagnosed as having NCSE. In 4 patients, a bilateral intraventricular hemorrhage was observed. Evaluation of persistent coma or neurological deterioration was the reason for which continuous EEG monitoring was carried out. Despite the fact that NCSE was controlled in 5 cases, none of those subjects experienced a significant clinical improvement and all

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**Figure 1.**

A CT scan of the brain revealed the existence of a large intraventricular hemorrhage occupying the left ventricle.

**Figure 2A.**

An EEG performed 72 hours after admission showed the presence of continuous epileptiform discharges (arrow) involving the entire left hemisphere. Vertical bar: 100 μ V; Horizontal bar: 1 second; Low filter: 0.5. High filter: 30 Hz.

of them died. Similarly, a sudden neurological worsening was the reason for performing an EEG in our case, and NCSE was refractory to conventional antiepileptic treatment. Moreover, NCSE remained uncontrolled for several days, and EEG was the unique method to demonstrate the persistence of ongoing epileptic activity.

It could be argued that in our case the long period of obtundation may have simply been related to the intraventricular hemorrhage and the electroencephalographic anomalies constituted only an epiphénoménon. Nevertheless, the behavioral improvement was closely associated with the abolition of the epileptiform activity on

**Figure 2B.**

On the next days, several EEGs revealed generalized continuous epileptiform activity in keeping with a secondarily generalized NCSE. Vertical bar: 100 µV; Horizontal bar: 1 second; Low filter: 0.5. High filter: 30 Hz.

**Figure 2C.**

An EEG carried out 23 days after admission showed findings in keeping with a diffuse encephalopathy with resolution of NCSE, but with some scattered spikes maximal on the right anterior temporal region. A significant improvement of the neurological state of the patient was observed at that time. Vertical bar: 100 µV; Horizontal bar: 1 second; Low filter: 0.5; High filter: 30 Hz.

the EEG, suggesting strongly that clinical symptoms had a genuine epileptic origin. Some speculation exists in relation to the pathophysiologic mechanism originating the NCSE in this case. Likely, the small hematoma localized in the left thalamus could be the epileptogenic source of the ipsilateral hemispheric continuous epileptiform discharges. However, an intrinsic epileptogenicity associated with the intraventricular hemorrhage *per se*, or acute hydrocephalus through disruption of cortico-subcortical pathways, may not be completely ruled out. Thus, it is well-known that the existence of seizures and pathological EEG features in neonates with severe intraventricular hemorrhage are considered markers of brain injury. Our patient had also a previous history of an ipsilateral middle cerebral artery territory infarction, which could have predisposed the occurrence of seizures from left hemispheric origin.

Prolonged localization-related (partial) NCSE is a harmless epileptic condition, which may cause, particularly

in the setting of acute brain disorders, permanent brain damage.⁴⁻⁶ Therefore, an early diagnosis and rapid treatment will avoid additional deficits. Although the prognosis of our patient was more favorable than described by Dennis et al.,³ the clinical improvement was markedly slow and neurological outcome poor. Unfortunately, it is not possible to perform continuous EEG monitoring in critically ill patients with altered mental status in our hospital. However, we are aware that this rapidly expanding electro-physiologic technique is the method of choice for evaluating subjects with cerebral damage and unexplained persistent confusion or coma.⁷

To summarize, NCSE is a possible severe neurological complication in older patients suffering from intraventricular hemorrhage. Since an unexplained deterioration of consciousness level may be the sole clinical sign of NCSE in these subjects, a high level of suspicion and an urgent EEG will be the cornerstone of an early and accurate diagnosis.

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